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LEFT HEMIPLEGIA

Followed by Loss of the Deep and Superficial Reflexes, Considerable Muscular Atrophy, Marked Anæsthesia in the Distal Portions of the Limbs, Loss of Faradaic Irritability, and Reaction of Degeneration of the Muscles on the Paralyzed Side.

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WITH REMARKS BY

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Report of the Case by Dr. Eskridge.

The trophic disturbances found in the case which is reported in this paper differ so widely from those that usually occur in old hemiplegias of brain origin, that a full record of all the symptoms is demanded.

Some years ago Eisenlohr cited two cases of cerebral palsy in which atrophy and degenerative reaction occurred, and recently he has had opportunity to make autopsies in both of his cases, finding degeneration of the peripheral nerves to account for the trophic changes in the affected muscles (F. Peterson, *Journal of Nervous and Mental Diseases*, April, 1894, p. 263). Dr. C. A. Herter states that "in one case coming under his observation a woman aged

seventy years, the patient had a series of slight apoplectic attacks, succeeded in time by complete paralysis of the right side of the body, face, arm, and leg, and complete motor aphasia; at the end of a week distinct atrophy of the muscles of the forearm was noticeable, and at the end of a month was very pronounced. The faradaic irritability of certain muscles of the forearm was very much reduced. The contractions were exceedingly sluggish, and the reaction of degeneration was undoubtedly present. The galvanic irritability of these muscles was somewhat diminished" (*ibid.*, p. 257). The literature relating to muscular degeneration following cerebral palsy in the adult is exceedingly meager, but the cases that are on record have not been made public in journals to which I at present have access. To make this case the more interesting, I have asked my friend, Dr. Frederick Peterson, of New York, to add any comments that he may see fit, together with the literature of the subject.

The patient, a Swedish widow woman, forty-two years of age, domestic by occupation, came to Colorado directly from Sweden in 1886. The father died of heart disease. The mother died, aged forty-two years, but the cause of her death is unknown. Other portions of the family history are negative. With the exception of an attack of measles when a small child, the patient enjoyed excellent health until she suffered from small-pox nine years ago. She apparently made a complete recovery from the latter and remained well for two years. In 1888, after having been exposed to the sun's rays, she suffered severely for some days from a headache and physical depression; but these symptoms soon passed off and she again felt as well as usual. In January, 1891, she had an attack of "the grip," and on the morning of January 25th, after convalescence had fairly set in, she was sitting in a chair, when she experienced a peculiar numb sensation in the left side of the head, face, and throughout the left side of the body, including the arm and leg.

The feeling alarmed her, and she attempted to stand, but found that her left leg was paralyzed, and about the same time she noticed that the left arm and the left side of the face were paretic. She is sure that consciousness was not affected. Within a few hours after the first appearance of the paralysis she was able to walk with slight assistance. For the next two or three years there was not much change in her condition, with the exception that the paresis passed away in the face. About two months ago she experienced considerable pain in the left leg below the knee, with more or less pain in the left knee and ankle joints. Since then she does not think she has been able to walk as well as before. The same numb sensation that she experienced in the left arm and leg in January, 1891, when her disability from paralysis began, still persists. She presents no distinct evidence of syphilis, denies alcoholic indulgence, and says she has never been subject to headache. Her speech has not been affected by the brain lesion.

The patient was admitted to the Arapahoe County Hospital, May 22, 1894. Examination, May 25th. She is able to walk with the assistance of a cane, but locomotion is very difficult on account of extreme weakness of the left leg. She exhibits no ataxia in any position that she assumes, but she is unable to bear much weight on the left leg The flexors and extensors of the left ankle are completely paralyzed, and the only movement that she can exert with the left foot is slight flexion of the great toe. The extensors of the left knee are too weak to extend the leg when she is sitting, and the flexors of this joint are extremely weak. The flexors of the left hip are absolutely powerless, and the extensors almost so. All movements of the right leg and foot are normal and vigorous. Dyn. R. 120; L. 60. Flexors and extensors of the left hand are weak, those of the elbow fairly strong, and the muscles of the left shoulder are but little weaker than those of the right. All the muscles of the right hand and arm are strong. There is little or no perceptible weakness of the muscles of the left side of the face. The tongue is protruded in the median line. Knee jerks: R., increased; L., absent. Ankle clonus absent. Reflex of the tendo Achillis: R., absent; L., absent. Plantar

reflexes absent. Abdominal reflexes: R., present; L., absent. Reflexes of extensor muscles of forearms: R., increased; L., absent. Biceps: R., increased; L., lessened. Triceps about normal. Measurements in inches—Calf: R., 13½; L., 12⅛. Just above knee: R., 16; L., 14½. Forearm: R., 10⅞; L., 10. Biceps: lax—R., 12; L., 11½; contracted—R., 13½; L., 12⅜. All the muscles of the left leg are soft and nearly flaccid.

Tactile Sense.—She is unable to recognize the contact of a feather on the left leg from the knee downward, including the foot and ankle. Over the left thigh this sense is present, but much less acute than it is on the right side. Over the palmar surface of the left hand and the dorsal surface of the fingers tactile sense is nearly lost. It is also much impaired from the elbow downward, but not nearly so greatly as it is in the left leg. Over the entire left side of the trunk there is lessening of the acuity of tactile sense, with delay in rapidity of conduction of tactile impressions, but in no portion of the trunk, around the anus, or over the external genitalia, are there any spots of tactile anæsthesia. In the left arm from the elbow upward and over the left side of the neck, face, and head there is perceived a slight impairment of tactile sense; but this can only be recognized by comparing these areas with the corresponding ones on the opposite side. Tactile sense is normal on the right side. Temperature sense is perverted most markedly where tactile sense is most disturbed. Both warm and cool substances give an impression of cold at first. After warm substances have been in contact with the skin for several seconds she then says that they feel warm. There is a slight perversion with a delay of temperature sense throughout the left side of the body. Localization sense is nearly normal, but slightly perverted over the left foot and ankle. Pain sense is most perverted over the anæsthetic areas, but on no portion of the body is it absent. Pressure and joint senses normal. It is impossible to test muscular sense in the affected parts on account of the extreme degree of paralysis. All sensory phenomena are normal on the right side. Eyes: R. V. = $\frac{20}{40}$; L. V. = $\frac{20}{30}$. Pupils equal and react to light and accommodation. Fields normal, except slight narrowing of the nasal side of the left eye. No weakening of

the external ocular muscles. Fundi and discs nearly normal. There is no disturbance in taste, smell, and hearing. Muscular response to faradaic current (the large size Flemming battery was used). Tibialis anticus: R., I plus 2½; L. will not respond to the strongest current. Peroneus group: R., I plus 2½; L., no response to strongest current. Extensor longus digitorum: R., II; L., no reaction to strongest current. Posterior tibial group: R., I plus 4; L., III. Rectus femoris: R., I plus 2¾; L., no response to strongest current. Posterior thigh group: R., II plus 1¼; L., IV plus 1¾. Flexors of wrist: R., I; L., I plus 2. Extensors of wrist: R., I plus 2½; L., I plus 4½. Triceps: R., I plus ½; L., I plus 2. Biceps: R., I; L., II. Deltoid: R., I plus 3; L., I plus 3 Reactions to the galvanic current—Tibialis anticus: K Cl C, R., 5 mil.; L., 30 mil., no response. An O C, R., 6½ mil.; L., 30 mil., no response. Peroneus group: K Cl C, R., 4 mil.; L., 25 mil. An O C, R., 5 mil.; L., 20 mil. Extensor longus digitorum: K Cl C, R., 6 mil.; L., 30 mil., no response. An O C, R., 7 mil.; L., 30 mil., no response. Posterior tibial group: K Cl C, R., 6 mil.; L., 15 mil. An O C, R., 7 mil; L., 12 mil. Rectus femoris: K Cl C, R., 8 mil.; L., 30 mil., no response. An O C, R., 10 mil.; L., 30 mil., no response. Extensors of wrist: K Cl C, R., 4 mil.; L., 6 mil. An O C, R., 6 mil.; L., 7 mil. Flexors of wrist: K Cl C, R., 3½ mil.; L., 5 mil. An O C, R., 5½ mil.; L., 6 mil. Triceps: K Cl C, R., 4 mil; L., 4½ mil. An O C, R., 6 mil.; L., 6 mil. Biceps: K Cl C, R., 4 mil.; L., 4½ mil. An O C, R., 6 mil.; L., 6 mil. Deltoid: K Cl C, R., 5 mil.; L., 5 mil. An O C, R., 6½ mil.; L., 6 mil.

The cause of the hemiplegia in this case is probably thrombotic occlusion of the arteries supplying the right internal capsule, and the muscular atrophy with the electrical changes and loss of myotatic irritability have been the result of unilateral multiple neuritis. The case would possess greater interest if it had been under intelligent observation from the onset of the hemiplegic symptoms until the present. In the absence of a complete history it must

be uncertain whether the neuritis dates from the time near the occurrence of the cerebral paralysis or from March, 1894—a little more than three years subsequently, when she experienced considerable pain in the left leg below the knee with more or less pain in the left knee and ankle joints. She does not think she has been able to walk as well since March, 1894, as she was previously, and she does not know when the wasting of the left leg and arm began.

Remarks of Dr. Peterson.

In the great majority of cases of hemiplegia there is no atrophy of muscles on the affected side. Sometimes we note a slight diminution in the size of the limbs involved, but this is merely from inactivity and disuse. It is not a true trophic change.

In certain rare cases, however, the diminution in size is so marked as to merit the name of an actual atrophy. These cases, one of which is described above by Dr. Eskridge, are so rare that, out of many hundreds of hemiplegiacs observed by me, I can recall but three or four in which the paralysis was accompanied by amyotrophy. Darkschewitsch (1) found six out of fifty-nine cases of hemiplegia examined for this purpose presenting muscular atrophies.

The behavior of the muscles in electrical examination is of considerable interest, because reaction of degeneration has always been one of our most trusted means for differentiating the two kinds of paralysis due to lesions in the cortico-spinal and spino muscular portions of the motor tract. Now, in some of these cerebral amyotrophies there is a change in the electrical reactions, but this change must be looked upon as exceedingly rare even in these cases. As a rule, we shall find perfectly normal reactions as re-

gards quantity and quality, whether to faradism or galvanism, in these cases of cerebral amyotrophy. The muscular atrophies from cerebral lesions behave in this respect very much like atrophies from joint lesions, the so-called arthropathic amyotrophies. But in a certain small minority of cases we may find some electrical changes, usually quantitative, very seldom qualitative.

As long ago as 1887 Wernicke (2) called attention to the diminution of faradaic and galvanic irritability on the paralyzed side in hemiplegia with hemianæsthesia. Even before that, Erb, in his book on electricity, had noted the occasional occurrence of diminished excitability in hemiplegic cases. Quincke (3) found very slight diminution of electrical irritability in his cases. In the six cases of Darkschewitsch the electric reactions were normal in four, and there was merely a quantitative reduction to faradism and galvanism in the other two cases. Eisenlohr (4) reported both quantitative and qualitative changes—in fact, complete reaction of degeneration—in his first case.

It is therefore proper to conclude that a reaction of degeneration in a case of cerebral amyotrophy is an extremely rare phenomenon.

What is perhaps of greater interest and importance in such cases is the origin of the atrophy. This is still a mystery, and one well worth solving. Various theories have been advanced to explain it, such as the following:

1. That there are trophic centers in the motor cortical area (Quincke).

2. That there are changes in the ganglion cells of the anterior horns (Charcot, Leyden, Brissaud (5), Joffroy, and Achard).

3. That the atrophy is due to peripheral neuritis (Déjérine, Eskridge).

4. That the wasting is explicable on the ground of defi-

cient nutrition due to purely vaso motor changes and diminished circulation (Roth and Muratow).

5. That there are functional disturbances in the spinal trophic centers due to dynamic influences from the lesion above.

6. That there are functional disturbances in the spinal trophic centers due to dynamic influences of a reflex kind from below along sensory paths.

Now, as to the results of autopsies in such cases, there are but few good data at hand.

As to the *cerebral lesions* in such cases, they vary in site, but are mostly about the internal capsule and optic thalamus.

There is usually descending degeneration of the *pyramidal tract* involved.

The ganglion cells of the *anterior horns* were found normal in one case by Quincke, in two by Eisenlohr, in one by Steiner (6), and in one by Darkschewitsch. The ganglion cells of the anterior horns were affected in two cases reported by Joffroy and Achard (7).

The *anterior roots* were examined by Darkschewitsch and Steiner in their cases and were normal.

The *peripheral nerves* were normal in all the cases where they were examined. Positive statements to this effect are made by Darkschewitsch and Steiner. Eisenlohr found slight simple degeneration in some of the peripheral nerves. This should not be called a neuritis.

The *muscles* themselves, examined by Darkschewitsch, presented a simple, not a degenerative, atrophy.

Thus we may say that in four of the six autopsies here described there were none of the anatomical changes in the spino-muscular portion of the motor tract usually incident to amyotrophy. This is corroborated by four careful observers. The joint research of Joffroy and Achard, in which

they describe changes in the anterior horns, stands alone in this respect.

It is worth while to consider, in connection with the cerebral amyotrophies of adults, the enormous retardation in growth of the affected extremities in infantile cerebral palsies. The paralyzed members grow, of course, as the child waxes in years, but the striking difference between the two sides can be explained only in one of two or three ways: Either there are trophic centers in the brain affected by the lesion, or the cerebral disorder influences dynamically the trophic centers in the spinal cord, or the retardation in growth may be due to vaso motor disturbances and insufficient blood-supply on the side involved.

A peculiar feature, too, of the cerebral amyotrophies of adults to which Darkschewitsch has called attention is the occurrence of actual arthropathies in many cases of hemiplegia, and the coincident muscular atrophies in some of such cases may be considered as joint atrophies after all. It will be noted that the case recorded by Dr. Eskridge presented evidences of arthropathic conditions in the knee and ankle joints of the paralyzed side.

Altogether, then, we may infer that there are as yet no conclusive data upon which to found a tenable theory as to the origin of muscular atrophy in hemiplegia.

A clinical fact worth remembering is that, as a rule, the amyotrophy comes on very rapidly and early after the onset of the hemiplegia, being sometimes noteworthy within two days.

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NOTE.—Since this article was set up an interesting case of cerebral amyotrophy from a meningo-cortical tumor has been recorded in the February number of the *American Journal of the Medical Sciences.*

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